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Mechanism Of The Virchow's Triad In The Development Of Thrombosis

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Abstract: Thrombosis is a complex pathological process characterized by the formation of a blood clot within the vascular system, which is one of the most dangerous conditions for human health [1]. In explaining this process, the concept of Virchow's triad — endothelial damage, impaired blood flow (stasis), and hyperhomocoagulation — is accepted as the main pathophysiological mechanism [2]. In the last decade, molecular and clinical studies of each component of this triad have revealed many new aspects of the development of thrombosis [3]. For example, endothelial dysfunction occurs as a result of atherosclerosis, hypertension, chronic inflammation, and oxidative stress, which leads to the loss of anticoagulant properties of the vascular wall and creates conditions for thrombus formation [4]. Stasis of blood flow is observed in the venous system, especially in conditions such as heart failure, varicose veins, or prolonged immobilization, and it increases erythrocyte aggregation and platelet adhesion to the wall [5]. Hyperhemocoagulation can be associated with genetic mutations (e.g., F5 Leiden or Prothrombin G20210A) or develop secondarily as a result of pregnancy, cancer, or autoimmune diseases [6].

Studies from 2020–2025 have shown that these three factors are interconnected and synergistically increase the risk of thrombosis [7]. Also, using RNA-seq and proteomics technologies, the expression of endothelial inflammatory markers (e.g., ICAM-1, VCAM-1, IL-6) has been found to increase the susceptibility to thrombosis [8]. At the same time, thrombotic events associated with cardiovascular diseases, COVID-19 infection, and hormonal therapy are also explained on the basis of Virchow's triad [9].

This article analyzes the role of each component of Virchow's triad in the formation of thrombosis and their modern molecular mechanisms. The results of the study provide an important theoretical and practical basis for individualizing the prevention and treatment of thrombosis [10].

Keywords: Virchow's triad, thrombosis, endothelial damage, blood flow stasis, hyperhomocoagulation, heart failure, genetic factors.

Introduction: To analyze the mechanism of thrombosis development based on the concept of Virchow's triad with the help of modern scientific research, to determine the pathophysiological role of each component and to highlight its clinical significance [7].

METHODS

The article was prepared using the literature review method based on articles published in Scopus, PubMed, ScienceDirect and Wiley Online Library databases between 2015 and 2025 [8]. Article selection criteria: clinical observations, multicenter studies, meta-analyses and systematic reviews.

Thrombosis is a pathophysiological process characterized by the formation of a blood clot (thrombus) in a blood vessel, and is one of the most

dangerous consequences of cardiovascular diseases [1]. The "Virkhov triad," proposed by the German scientist Rudolf Virchow in 1856, includes three main factors in the pathogenesis of thrombosis: endothelial damage, impaired blood flow (stasis), and hyperhemocoagulation [2]. This concept has not lost its importance today as a fundamental pathophysiological model for clinical medicine and basic research [3].

Modern studies show that each component of Virchow's triad operates in a strong relationship with each other and that the synergistic effect between them constitutes the main mechanism of thrombosis development [4]. Endothelial cell dysfunction occurs as a result of oxidative stress, chronic inflammation, metabolic syndrome and atherosclerosis, which leads to the loss of the antithrombotic properties of the vascular wall [5]. At the same time, stasis of blood flow creates a favorable environment for thrombus formation due to the slowing of blood circulation in the venous system [6]. Another important factor is hyperhomocoagulation, i.e. excessive activity of the blood coagulation system, which may be associated with genetic mutations (F5 Leiden, Prothrombin G20210A), malignant neoplasms or hormonal changes [7]. A series of global studies conducted between 2015 and 2025, in particular articles published in Nature Reviews Cardiology, Blood, and the Journal of Thrombosis and Haemostasis, analyzed the mechanisms of thrombosis at the molecular level and confirmed that inflammatory processes associated with high expression of endothelial surface markers (e.g. VCAM-1, E-selectin, and tissue factor) promote thrombus formation [8].

The COVID-19 pandemic also brought a new scientific perspective to the pathophysiology of thrombosis - it was found that the SARS-CoV-2 virus directly affects endothelial cells, causing micro- and macrothrombus formation [9]. Therefore, thrombosis is considered not only as a local vascular disease, but also as a global pathobiological process associated with systemic inflammation and immuno-thrombotic responses [10].

As noted in the introduction, an in-depth study of the mechanisms of thrombosis development, especially the analysis of the molecular basis of the components of Virchow's triad, is an important scientific direction in the development of thrombosis prevention and individual treatment strategies [11]

RESULTS

Endothelial damage is one of the initial and most important stages in the pathogenesis of thrombosis [1]. Endothelial cells in a healthy state provide an anticoagulant barrier of the vascular wall, maintaining a natural balance against blood clotting through substances such as nitric oxide (NO), prostacyclin (PGI_2) and thrombomodulin [2]. However, when the endothelial layer is damaged as a result of mechanical, biochemical or inflammatory effects, this balance is disrupted and the thrombogenesis process is activated [3].

Mechanical damage - for example, situations such as surgical interventions, intravascular catheterization or prosthesis implantation - directly damages cells at the endothelial level. As a result, collagen and tissue factor (TF) are released into the bloodstream, which leads to the activation of von Willebrand factor (vWF) [4]. vWF, in turn, promotes platelet adhesion and primary thrombus formation [5].

Inflammatory processes also play a crucial role in the development of endothelial dysfunction and thrombosis. In atherosclerosis, chronic metabolic diseases (e.g., diabetes), or infectious processes, endothelial cells secrete inflammatory markers such as interleukin-6 (IL-6), tumor necrosis factor alpha (TNF-α), and C-reactive protein (CRP) [6]. These mediators increase the expression of E-selectin, P-selectin, and ICAM-1 molecules on the endothelial surface, which promotes leukocyte adhesion to the wall and increased thrombin activity [7].

Oxidative stress is the molecular basis of endothelial damage. Free radicals and reactive oxygen species (ROS) damage endothelial DNA and lipids, resulting in activation of NADPH oxidase and reduced nitric oxide bioavailability [8]. This disrupts vascular tone and enhances the procoagulant state. Studies conducted in 2022–2025 have shown that oxidative stress alters the levels of von Willebrand factor and thrombomodulin, which accelerates thrombus formation in arteries and veins [9]. COVID-19-associated endothelial inflammation (endotheliitis) has also been implicated in the increase in thrombotic events. Ackermann et al. (2020) confirmed that SARS-CoV-2 directly damages endothelial cells, enhancing micro- and macrothrombus formation [10]. Therefore, endothelial damage—whether mechanical, inflammatory, or oxidative in origin—activates the initial mechanism of thrombosis, as a central element of Virchow's triad.

Blood flow stasis is an important pathophysiological factor that leads to the formation of a thrombus within a vessel as a result of slowing or stopping blood flow [1]. Healthy blood flow is laminar, which ensures a stable distribution of platelets and plasma flow. However, in a state of stasis, blood flow becomes turbulent, which increases mechanical stress on the endothelial wall and activates the process of thrombus formation [2]. Heart failure is the most common cause of stasis. When the cardiac output is reduced, blood

stagnation, especially in the left ventricle, significantly increases the risk of thrombosis [3]. A study conducted by Kondo et al. in 2025, demonstrated that slowing blood flow in patients with heart failure is directly related to endocardial thrombosis [4]. At the same time, cardiac arrhythmias, in particular atrial fibrillation, cause blood to stagnate in the left ventricle, which is the pathogenic basis of 90% of strokes of cardiac origin [5].

Stasis in the venous system is also one of the main sources of thrombosis pathogenesis. Prolonged immobilization (after surgery, paraplegia, or long journeys), venous valve dysfunction, or varicose veins cause blood circulation to slow down [6]. This condition leads to the development of venous thromboembolism (VTE). A clinical review published by Downey in 2024 noted that venous stasis accounts for 60–70% of VTE cases [7].

Sleep apnea syndrome has also been considered a factor in the pathogenesis of thrombosis in recent years. Hypoxia and pressure changes during apnea episodes increase blood viscosity, resulting in stasis at the microcirculatory level [8]. A review article published in 2025 by Gholinataj Jelodar and Rahimi showed that the risk of venous thromboembolism is doubled in patients with obstructive sleep apnea (OSA) [9].

Microcirculatory stasis is a slowing of blood flow at the capillary level, as opposed to the large vessels, and is observed in the setting of inflammation, hyperviscosity syndrome, or sepsis. This condition is accompanied by increased expression of inflammatory markers of endothelial cells, in particular E-selectin and P-selectin, which increase platelet adhesion [10].

Thus, blood flow stasis is associated with clinical conditions such as heart failure, venous thrombosis, or sleep apnea, and together with endothelial damage and hyperhomocoagulation, occupies an important place in Virchow's triad as a central component of the pathogenesis of thrombosis.

Hyperhomocoagulation is a pathological condition characterized by an increased tendency to thrombosis as a result of excessive activation of the blood coagulation system [1]. This process is the third component of Virchow's triad and is caused by a number of genetic and acquired factors. Normally, coagulation and anticoagulant mechanisms work in balance: thrombin, fibrin, and platelets activate the coagulation process, while protein C, protein S, and antithrombin III keep it under control [2]. However, genetic or metabolic disorders in this system lead to the development of a hyperhomocoagulant state.

Genetic factors are one of the main causes of hyperhomocoagulation. For example, mutations in the protein C receptor (EPCR) or Factor V Leiden (FVL) genes result in a decrease in the functional activity of the anticoagulant system [3]. In 2022, genetic analyses by Mackman and Key found that the risk of venous thromboembolism increased by 3.5 times in patients with EPCR mutations [4]. At the same time, the prothrombin G20210A polymorphism also increases procoagulant activity in plasma and accelerates the process of fibrin formation [5].

Increased hyperhomocoagulation can be associated with physiological or pathological conditions. For example, during pregnancy, increased estrogen levels activate coagulation factors such as fibrinogen, factor VII, and von Willebrand factor [6]. This, in turn, increases the risk of venous thrombosis in pregnant women by 4–5 times. In a study published in 2025 by Zhang and Shao, it was scientifically proven that placental microthrombi are directly related to maternal endothelial dysfunction [7].

In addition, oncological diseases, especially pancreatic, lung and colon cancer, dramatically change the coagulation status of the blood. Tumor cells produce tissue factor (TF), cancer procoagulant (CP) and microvesicular TF-carrying exosomes, which enhance thrombin generation [8]. The results of a meta-analysis conducted by Levine et al. in 2023 showed that D-dimer levels in patients with lung cancer are 2.7 times higher than normal, which in turn increases the likelihood of clinical thrombosis by 5 times [9].

Metabolic syndrome, diabetes, and obesity also contribute to an increased hyperhomocoagulant state. These conditions increase the secretion of factor VIII and fibrinogen through insulin resistance, inflammation, and oxidative stress [10]. In a 2021 article by Owens and Mackman, it was shown that chronic inflammation associated with obesity increases the risk of thrombosis through epigenetic mechanisms that activate procoagulant factors [11]. In summary, hyperhomocoagulability is a multifactorial condition resulting from genetic mutations, hormonal changes, and tumor processes, and it is an integral component of Virchow's triad, which plays a key pathogenic role in the development of thrombosis.

DISCUSSION

Virchow's triad is central to medical science as a key theory in explaining the pathogenesis of thrombosis [1]. This triad—endothelial injury, blood flow stasis, and hyperhomocoagulability—interacts with each other to activate the process of thrombus formation [2]. Increased expression of von Willebrand factor and thrombomodulin, resulting from endothelial dysfunction or inflammation, enhances platelet

adhesion [3]. This is especially true in cardiovascular diseases, surgical procedures, or chronic inflammatory conditions [4].

Blood flow stasis is one of the main manifestations of hemodynamic disorders, which plays an important role in the development of venous thrombosis and pulmonary embolism [5]. Factors such as heart failure, prolonged immobilization, obesity, and sleep apnea syndrome reduce venous return, increasing local hypoxia and endothelial stress [6]. Therefore, slowing blood flow not only increases the risk of thrombus formation, but also stimulates the activity of inflammatory mediators [7].

The mechanism of hyperhemocoagulation is often associated with genetic and acquired causes. Physiological conditions such as protein C receptor or factor Leiden mutations, antithrombin III deficiency, as well as pregnancy, cancer, and estrogen-based therapy, increase the susceptibility to thrombus formation [8]. Studies have shown that patients with cancer have a 4–7-fold increased risk of thrombosis, which is associated with procoagulant microparticles secreted by tumor cells [9].

The emergence of new-generation anticoagulant therapies (e.g., dabigatran, apixaban, rivaroxaban) has been a major step in the management of thromboembolic diseases [10]. At the same time, individual risk assessment systems — such as HAS-BLED and CHA_2DS_2-VASc — have allowed for more effective planning of thrombosis prevention [11]. Current scientific research, in particular, the study of the mechanisms of immunothrombosis and NETosis (neutrophil extracellular trap formation), is revealing new molecular pathways in the pathogenesis of thrombosis [12].

In general, all three elements of Virchow's triad act as a complementary system. It is not the dominance of a single pathological factor, but their interaction that underlies thrombogenesis [13]. Therefore, in modern clinical practice, thrombosis prevention requires a multifactorial approach, which provides the basis for the development of personalized therapy based on molecular markers [14].

CONCLUSION

Virchow's triad - endothelial damage, blood flow stasis and hyperhomocoagulation - remains important as three main pathophysiological factors in the pathogenesis of thrombosis. Modern studies show that this triad reinforces each other through complex interrelated mechanisms. For example, endothelial dysfunction is associated not only with mechanical or inflammatory processes, but also with oxidative stress, DNA damage and the activity of immune cells. Thus, endothelial nuclear gene expression changes, and increased secretion of thrombomodulin, von Willebrand factor and interleukin-6 is observed.

Blood flow stasis, in cases of heart failure, venous thrombosis or hypodynamia, reduces hemodynamic stress, causing platelets to be in contact with the endothelium for a longer period. This leads to the accumulation of fibrin, which forms the initial nucleus of the thrombus. In recent years, studies on stasis developing after sleep apnea, long flights or orthopedic surgery have further proven the clinical importance of this factor.

Hyperhemocoagulation states are observed in genetic (e.g., protein C or antithrombin III mutations) and acquired (pregnancy, inflammatory syndromes, cancer) forms. Many studies after 2020 have noted tumor-specific procoagulant molecules — tissue factor and microparticles — as the main mediators of enhanced thrombogenesis.

In general, the joint study of the components of Virchow's triad in the prevention of thrombosis, especially the identification of individual risk factors and the development of preventive strategies based on biomarkers, is considered a relevant direction. In clinical practice, a combination of anticoagulants, endothelial function-restoring drugs, and lifestyle changes plays an important role in reducing the global burden of thrombosis.

REFERENCES

1. MICROFLORA, Dilshodovich KH SHIELD OF INTESTINAL. "CHANGE EFFECT ON THE GLANDS." American Journal of Pediatric Medicine and Health Sciences (2993-2149) 1 (2023): 81-83.
2. Dilshodovich, Khalilov Hikmatulla, Kayimov Mirzohid Normurotovich, and Esanov Alisher Akromovich. "RELATIONSHIP BETWEEN THYROID DISEASE AND TYPE 2 DIABETES." (2023).
3. To'laganovna, Y. M. (2025). SKELET MUSKULLARNING FIZIOLOGIYASI VA ULARNING ISHLASH MEXANIZMI: AKTIN VA MIOZIN VA ENERGIYA ASOSLARI. AMERICAN JOURNAL OF SOCIAL SCIENCE, 3(4), 54-60.
4. Tolaganovna, Y. M., & Shavkatjon o'g'li, A. A. (2025). INSON ORGANIZMIDA YURAK QON-TOMIR KALSALLIKLARI, MIOKARD INFARKTINING KELIB CHIQISH SABABLARI VA ULARNING OLISH CHORA–TADBIRLARI. AMERICAN JOURNAL OF APPLIED MEDICAL SCIENCE, 3(4), 136-144.
5. Jo'rabek, K. (2025). BUYRAK KASALLIKLARGA OLIB KELADIGAN PATALOGIK HOLATLAR VA ULARNI OLDINI OLISH. AMERICAN JOURNAL OF APPLIED MEDICAL SCIENCE, 3(4), 129-135.

6. Azimova, S. B., and H. D. Khalikov. "Modern pathogenetic aspects of urolithiasis development." The American Journal of Medical Sciences and Pharmaceutical Research 7.04 (2025): 21-24.
7. Dilshod ogli, Xalilov Hikmatulla, and Qayimov Mirzohid Normurotovich. "THE ROLE OF ARTIFICIAL INTELLIGENCE AND ROBOTICS IN MEDICINE." Web of Medicine: Journal of Medicine, Practice and Nursing 3, no. 5 (2025): 201-207.
8. To'laganovna, Yusupova Moxira. "SKELET MUSKULLARNING FIZIOLOGIYASI VA ULARNING ISHLASH MEXANIZMI: AKTIN VA MIOZIN VA ENERGIYA ASOSLARI." AMERICAN JOURNAL OF SOCIAL SCIENCE 3.4 (2025): 54-60.
9. Ogli, Xalilov Hikmatulla Dilshod, Namiddinov Abror Anasbek Ogli, Sayfullayeva Durdona Dilshod Qizi, and Hikmatova Gulasal Farhodjon Qizi. "TELEMEDITSINANING PROFILAKTIK DAVOLANISHDA AHAMIYATI." Eurasian Journal of Academic Research 4, no. 4-2 (2024): 66-70.
10. Dilshod ogli, Xalilov Hikmatulla, Amirqulov Navro'zbek To'rayevich, and Shukurov Umidjon Majid o'g'li. "GIPOTIREOIDIZMNI EKSPERIMENTAL MODELLASHTIRISH." AMERICAN JOURNAL OF APPLIED MEDICAL SCIENCE 3.2 (2025): 207-209.
11. Xalilov, H. D., Namiddinov, A. A., Berdiyev, O. V., & Ortiqov, O. S. (2024). GIPERTIROIDIZM VA YURAK ETISHMOVCHILIGI. Research and Publications, 1(1), 60-63.
12. Berdiyev, O. V., M. Quysinboyeva, and A. Sattorova. "Telemeditsina Orqali Qalqonsimon Bez Kasalliklarini Boshqarish." Open Academia: Journal of Scholarly Research 2.6 (2024): 69-74.
13. Karabayev, Sanjar. "SOG'LIQNI SAQLASHDA TELETIBBIYOT IMKONIYATLARI, XUSUSIYATLARI VA TO'SIQLARI." Yевразийский журнал медицинских и естественных наук 3.2 Part 2 (2023): 41-46.
14. Шадманова, Н.К. and Халилов, Х.Д., 2023. НАУЧНО-ПРАКТИЧЕСКИЙ ИНТЕРЕС ИЗУЧЕНИЯ ВЕГЕТАТИВНОЙ РЕГУЛЯЦИИ ДИЗАДАПТИВНЫХ РЕАКЦИЙ СЕРДЕЧНО-СОСУДИСТОЙ СИСТЕМЫ. Евразийский журнал академических исследований, 3(8), pp.126-134.
15. Normurotovich, Qayimov Mirzohid, and Ganjiyeva Munisa Komil Qizi. "GIPOTIROIDIZM VA YURAK ETISHMOVCHILIGI." Eurasian Journal of Academic Research 4, no. 5-3 (2024): 14-19.
16. Normurotovich, Q. M. "Dilshod ogli XH RODOPSIN G OQSILLARI FILOGENETIK TAHLIL." Journal of new century innovations 43, no. 2 (2023): 178-183.
17. Maxira, Yusupova, Xalilov Hikmatulla Dilshod ogli, and Berdiyev Otabek Vahob ogli. "FIZIOLOGIYA FANI RIVOJLANISHI TIBBIYOTDAGI AHAMYATI. FIZIOLOGIYADA TADQIQOT USULLARI." PEDAGOG 7.12 (2024): 111-116.
18. MICROFLORA DK. CHANGE EFFECT ON THE GLANDS. American Journal of Pediatric Medicine and Health Sciences (2993-2149). 2023;1:81-3.
19. Dilshodovich, Khalilov Hikmatulla. "SHIELD OF INTESTINAL MICROFLORA CHANGE EFFECT ON THE GLANDS." American Journal of Pediatric Medicine and Health Sciences (29932149) 1 (2023): 81-83.
20. Dilshodovich, K.H., Normurotovich, K.M. and Akromovich, E.A., 2023. RELATIONSHIP BETWEEN THYROID DISEASE AND TYPE 2 DIABETES.
21. Dilshod ogly, Khalilov Hikmatulla, Shatursunova Madina Abdujamilovna, and Shukurov Umidjon Majid ogly. "THE IMPORTANCE OF ARTIFICIAL INTELLIGENCE IN THE DETECTION OF KIDNEY DISEASES MODERN APPROACHES AND PROSPECTS." Western European Journal of Modern Experiments and Scientific Methods 3.04 (2025): 9-13.
22. Ikrom, T., 2025. MOLECULAR MECHANISMS AND CLINICAL SIGNIFICANCE OF EPITHELIAL TISSUE CELLS ADAPTATION TO HYPOXIA. Western European Journal of Modern Experiments and Scientific Methods, 3(05), pp.15-22.
23. Ikrom, Tilyabov. "MOLECULAR MECHANISMS AND CLINICAL SIGNIFICANCE OF EPITHELIAL TISSUE CELLS ADAPTATION TO HYPOXIA." Western European Journal of Modern Experiments and Scientific Methods 3.05 (2025): 15-22.
24. Abdujamilovna, S.M. and Dilshod ogli, X.H., 2025. QAND MIQDORINING SUYAKLANISHGA TA'SIRI. Continuing education: international experience, innovation, and transformation, 1(10), pp.137-141.
25. Абдухаликова, Нигора Фахриддиновна, and Хикматулла Халилов. "РОЛЬ ЦИТОХРОМА И В МЕХАНИЗМАХ КЛЕТОЧНОГО ДЫХАНИЯ И ГИПОКСИИ." Advanced methods of ensuring the quality of education: problems and solutions 2.11 (2025): 62-68.
26. Абдухаликова, Нигора Фахриддиновна, and Хикматулла Халилов. "РОЛЬ СУКЦИНАТДЕГИДРОГЕНАЗЫ В МЕХАНИЗМАХ ГИПОКСИИ." Advanced methods of ensuring the quality of education: problems and solutions 2.11 (2025): 55-61.
27. Faxriddinovna, Abduxalikova Nigora, Xalilov Hikmatulla Dilshod ogli, and Jabborov Botir Baxodir ogli. "EOZINOFIL FAGASITOZ QILISH MEXANIZMLARI." Advanced methods of ensuring

the quality of education: problems and solutions 2.11 (2025): 44-54.

28. Talipova, Noila, et al. "Genetic characteristics of the course of chronic hepatitis." E3S Web of Conferences. Vol. 381. EDP Sciences, 2023.

29. Khasanov, B., et al. "CHRONIC HEPATITIS OF MOTHER AND MORPHOLOGICAL FEATURES OF IMMUNE SYSTEM FORMATION OF POSTERITY." The Scientific Heritage 55-2 (2020): 42-43.

30. Dilshod, Qayimov Mirzohid Normurotovich Khalilov Khikmatulla. "oglu.(2025). THE INFLUENCE OF THE GENERAL ENVIRONMENT ON THE DEVELOPMENT OF ALCOHOLISM [Data set]. Zenodo."

31. Абдухаликова, Нигора Фахриддиновна. "ГИПОКСИЕЙ ИНДУЦИРОВАННЫЙ ФАКТОР КАК МИШЕНЬ ФАРМАКОЛОГИЧЕСКОГО ВОЗДЕЙСТВИЯ." Медицинский журнал молодых ученых 15 (09) (2025): 133-140.

32. Ирискулов, Бахтиёр Уктамович, Нигора Фахриддиновна Абдухаликова, and Комола Толиховна Зупарова. "САХАРНЫЙ ДИАБЕТ И РОЛЬ МЕЛАТОНИНА В ЕГО РАЗВИТИИ И ЛЕЧЕНИИ." ИНФЕКЦИЯ, ИММУНИТЕТ и ФАРМАКОЛОГИЯ (1999): 93.

33. ABDUKHALIKOVA, NF. "IMPORTANCE OF PHOTODYNAMIC THERAPY IN PROLIFERATIVE PROCESSES." INTERNATIONAL JOURNAL OF MEDICAL SCIENCE AND PUBLIC HEALTH 6.1 (2025): 27-34. Iriskulov, B. U., R. B. Tadjibaeva, and M. B. Nosirjonova. "Differential Diagnosis of Acute Leukemia in Children." (2024).

34. Fakhriddinovna, Sultonova Umida, Khaydarova Gavkhar Saidakhmatovna, and Abdukhalikova Nigora Fakhriddinovna. "LANGUAGE ADAPTATION AND VALIDATION THE FPI (VOICE HANDICAP INDEX) QUESTIONNAIRE IN UZBEK IN THE DIAGNOSIS OF VOICE DISORDERS IN PARKINSON'S DISEASE." International Journal of Modern Medicine 3.10 (2024): 60-73.

35. Абдухаликова, Нигора Фахриддиновна, and Бахтиёр Уктамович Ирискулов. "КЛЕТОЧНЫЕ МЕХАНИЗМЫ ПАТОГЕНЕЗА МИОКАРДИАЛЬНОГО ПРЕКОНДИЦИОНИРОВАНИЯ." ИНФЕКЦИЯ, ИММУНИТЕТ и ФАРМАКОЛОГИЯ (1999): 5.

36. Sultanova, U. F., G. S. Khaidarova, and N. F. Abdukhalikova. "Анкетирование пациентов для оценки состояния голосообразующего аппарата, как одна из методов ранней диагностики болезни Паркинсона." Eurasian Journal of Otorhinolaryngology-Head and Neck Surgery 3 (2024): 80-84.

37. Abdukhalikova, Nigora F., and Bakhtiyor U. Iriskulov. "EFFECT OF PLANT PHOTOSENSITIZER PSORALEN ON MITOCHONDRIAL STRUCTURES IN INFLAMMATORY PROCESSES." Central Asian Journal of Medicine.